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Evaluation of body-mass index in patients with coronary artery ectasia

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ABSTRACT

Aims: Coronary artery ectasia (CAE) is defined as the dilation of a coronary artery to at least 1.5 times the diameter of the adjacent normal segment, detected in 0.3%-5.3% of coronary angiographies. The body-mass index (BMI), calculated as weight (kg) divided by height squared (m^2), is the simplest measure for diagnosing obesity. Obesity is linked to cardiovascular diseases, including CAE, type 2 diabetes, hypertension, and certain cancers, with higher BMI increasing the risk of disease. This study aimed to assess the BMI in patients with CAE and to investigate its potential association with the presence of the disease.

Methods: In our clinic, CAE patients diagnosed via coronary angiography were retrospectively evaluated, and their BMI values were compared. BMI was calculated using height and weight records from the hospital's automation system. The study included 111 patients and 111 control individuals.

Results: A total of 222 patients were enrolled in the study. It was observed that BMI values were significantly higher in individuals diagnosed with CAE compared to the control group. Moreover, several clinical parameters, including advanced age, diabetes mellitus, hypertension, smoking, and elevated low-density lipoprotein cholesterol (LDL) levels, were associated with the presence of CAE.

Conclusion: In this study, BMI was significantly higher in CAE patients. Obesity may contribute to CAE through metabolic factors. Further research is needed to clarify the mechanisms and assess the benefits of weight control in CAE prevention.

Keywords: Body-mass index (BMI), coronary artery ectasia (CAE), comorbid diseases

INTRODUCTION

Coronary artery ectasia (CAE) is a vascular anomaly characterized by the dilation of a coronary artery (CA) segment by at least 1.5 times or more compared to an adjacent healthy CA segment. CAE and CA aneurysms are detected in coronary angiographies at rates ranging from 0.3% to 5.3%. CAE is most commonly observed in the right CA, followed by the circumflex artery and the left anterior descending artery; respectively. The most common etiological cause of CAE is atherosclerosis, which accounts for approximately 50–60% of cases as the primary factor. Additionally, congenital anomalies (20–30%), inflammatory diseases (e.g., vasculitis), and connective tissue disorders (10–20%) are other significant contributors to the development of CAE. Although CAE is generally observed in conjunction with CA disease, it can also be detected in some individuals as an isolated condition.[1,2]

Diagnosis is typically established through invasive coronary angiography. Quantitative measurements enhance the objectivity of the evaluation and allow for more accurate determination of luminal dilation, particularly in terms of vessel diameter increase. In the literature, ectasia is classified based on diameter as small (<5 mm), moderate (5–8 mm), and large/"giant" (>8 mm).[3]

A recent article discusses the hemodynamic consequences of CAE, highlighting that the turbulent blood flow within ectatic arteries can lead to thrombus formation and distal embolization, which may impair myocardial perfusion. Consequently, the link between CAE and microvascular perfusion disorders strengthens, elevating the risk of myocardial perfusion insufficiency.[4] Although the pathophysiology of CAE is not yet fully understood, excessive vascular remodeling in response to atherosclerotic plaque growth is believed to play a significant role in disease

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progression. Genetic factors may also be involved in this process, warranting further investigation.[5] According to recent research, endothelial dysfunction and systemic inflammation are key factors in the pathophysiology of CAE. There is a substantial inflammatory component to the course of CAE, as evidenced by the raised levels of inflammatory markers such as interleukin-6 (IL-6) and high-sensitivity C-reactive protein (hs-CRP) in these patients.[6] The function of oxidative stress in patients with isolated CAE was examined in a study by Baysal et al.[7] While antioxidant markers showed no changes, it was discovered that CAE patients had considerably higher levels of lipid hydroperoxide, total oxidative status, and oxidative stress index. These results imply that elevated pro-oxidant activity might be a key factor in CAE development. The study further highlighted the connection between CAE and oxidative stress-related endothelial dysfunction and chronic inflammation. This lends credence to the idea that atherosclerosis and CAE may have comparable pathophysiological processes.[7]

Obesity is a key risk factor for many chronic diseases, especially cardiovascular disorders, and its rising prevalence has made it a global health concern. The body-mass index (BMI), which is determined by dividing body weight in kilograms by height in meters squared (kg/m^2), is the most widely used measure for identifying and categorizing obesity.[8] According to the World Health Organization (WHO) and other international guidelines, the classification based on BMI is as follows:

Normal BMI: 18.5–24.9 kg/m^2, Overweight: 25–29.9 kg/m^2, Obesity class 1 (mild obesity): 30–34.9 kg/m^2, class 2 (moderate obesity): 35–39.9 kg/m^2, class 3 (severe obesity, morbid obesity): ≥40 kg/m^2 (**Table 1**).[9] A high BMI increases the risk of developing cardiovascular diseases.[10] Beyond BMI classification, recent studies highlight the importance of visceral fat distribution in cardiovascular risk. Visceral adipose tissue, is strongly correlated with metabolic syndrome and atherosclerotic burden.[11] As a result, waist-to-hip ratio and waist circumference are now considered more precise indicators of cardiovascular risk than BMI alone.[12]

Table 1. Body-mass index scale

Who classification o weight status?	
Weight status	**Body-mass index (BMI), kg/m^2**
Underweight	<18.5
Normal range	18.5-24.9
Overweight	25.0-29.9
Obese	≥30
Obese class I	30.0-34.9
Obese class II	35.0-39.9
Obese class III	≥40

Obesity is directly associated with systemic diseases such as type 2 diabetes, hypertension, dyslipidemia, endothelial dysfunction and metabolic syndrome, all of which accelerate atherosclerotic processes and contribute to the development of CAE. Increased BMI is linked to insulin resistance, endothelial dysfunction, chronic inflammation, hyperinsulinemia, and elevated prothrombotic factor levels, all of which promote atherosclerotic changes in coronary arteries. Studies indicate that cardiovascular morbidity and mortality increase in individuals with a BMI exceeding 25 kg/m^2.[13,14]

Emerging evidence suggests that lifestyle interventions, such as dietary modifications and structured physical activity, may reduce cardiovascular risk by improving endothelial function and metabolic health.[15]

Understanding the potential relationship between CAE and BMI is crucial for better risk assessment and for evaluating the impact of obesity management on CAE development. Despite its rarity, CAE is a cardiovascular condition with significant clinical importance, and many aspects of its etiology, pathophysiology, and management remain unclear.[16]

This study aims to analyze the BMI values of patients diagnosed with CAE, assess the potential association between obesity and CAE, and assess the BMI in patients with CAE and to investigate its potential association with the presence of the disease. The findings of this research could enhance the understanding of CAE risk factors and highlight the critical role of obesity management not only for overall health but also for cardiovascular protection. Additionally, early BMI regulation and control of metabolic risk factors in individuals with CAE may be considered a potential strategy for preventing disease progression. Ultimately, this study seeks to provide new insights into the etiology and of CAE while laying the groundwork for future comprehensive research in this field. In this study, we will test the hypothesis of what kind of difference there will be in BMI in patients with CAE.

METHODS

The study was conducted with the permission of Bolu Abant İzzet Baysal University Non-interventional Clinical Researches Ethics Committee (Date: 21.01.2025, Decision No: 2025/23). All procedures were carried out in accordance with the ethical rules and the principles of the Declaration of Helsinki. Patients who were diagnosed with CAE through coronary angiography at our hospital between 2019 and 2024 were retrospectively analyzed to assess their BMI and compare it between different groups. The BMI values were systematically calculated based on the height and weight measurements recorded in the hospital's digital automation system, ensuring accuracy and consistency in the data collection process. The measurements were performed manually by different observers. However, all observers are physicians from our clinic, and standardization was ensured as the measurements were conducted based on a consensus established within our clinic. A total of 222 individuals were included in the study, evenly distributed between the patient group, consisting of individuals diagnosed with CAE, and the control group, composed of individuals without CAE. The control group was selected from individuals who were symptomatic, had a positive exercise test, met the exclusion criteria, and had normal coronary arteries as a result of coronary angiography.

To maintain the integrity of the study and eliminate potential confounding factors, strict exclusion criteria were applied. Patients diagnosed with severe valvular heart disease, advanced heart failure with an ejection fraction below 30%, severe acute or chronic liver and/or kidney failure, as well as those with advanced chronic lung disease, were excluded. Additionally, individuals under the age of 18 and patients experiencing severe systemic inflammation were not considered eligible for participation.

There is reasonable interobserver consistency in the diagnosis and classification of CAE, suggesting that experienced assessors interpret the condition similarly.

Statistical Analysis

The data analysis was conducted using SPSS software (SPSS 22.0 for Windows, IBM Corp., Chicago, IL, USA). Continuous variables were tested for normality using the Shapiro-Wilk test. Data with a normal distribution were presented as mean±standard deviation (SD) and compared using the independent samples t-test. Non-normally distributed variables were presented as median and interquartile range (IQR) and compared using the Mann–Whitney U test. Categorical variables were presented as percentages (%) and compared using the Chi-square (χ^2) test. The relationship between the existence of CAE and demographic and biochemical data was evaluated using Spearman correlation analysis. A p-value <0.05 was considered statistically significant.

RESULTS

Age was significantly higher in the CAE group compared to the control group (62.27±10.57 vs. 57.93±11.55 years, p=0.004). Weight was significantly higher in the CAE group (86.25±16.17 kg vs. 73.54±9.23 kg, p<0.001). Height did not differ significantly between the groups (p=0.208) (**Table 2**).

Table 2. Comparison of age, weight, and height between control and coronary ectasia groups

Variable	Control group	Coronary ectasia group	t	p
Age (years)	57.928±11.549	62.270±10.567	2.923	0.004
Weight (kg)	73.541±9.233	86.252±16.169	7.193	<0.001
Height (m)	1.682±0.067	1.695±0.087	1.264	0.208

kg: Kilogram, m: Meter, t: Student's t-test statistics

BMI was significantly higher in the CAE group (30.27±6.18 vs. 26.26±3.13, p<0.001). Also, total cholesterol (TCHOL) was significantly lower in the CAE group (p=0.012), while high-density lipoprotein cholesterol (HDL), triglyceride (TG), glucose, and hemoglobin A1c (HbA1c) levels did not show significant differences (p>0.05) (**Table 3**).

Gender distribution did not differ significantly between the groups (p=0.121). Smoking status did not show a statistically significant difference (p=0.785). Diabetes mellitus (DM) and hypertension (HT) prevalences were higher in the CAE group, but these differences did not reach statistical significance (**Table 4**).

Table 3. Comparison of BMI, lipid profile, and glucose levels between control and coronary ectasia groups

Variable	Control group	Coronary ectasia group	U	p
	Median-IQR	Median-IQR		
BMI (kg/m²)	26.260-3.130	30.274-6.182	9397.500	<0.001
HDL (mg/dl)	46.000-15.700	42.150-14.775	4449.000	0.094
LDL (mg/dl)	109.900-53.100	109.250-54.850	4424.500	0.051
TG (mg/dl)	146.000-112.250	125.500-77.750	4677.000	0.213
TCHOL (mg/dl)	198.000-63.000	182.000-61.250	4104.000	0.012
Glucose (mg/dl)	99.000-22.000	100.000-35.500	5892.000	0.265
HbA1c (%)	5.650-1.125	5.850-1.275	2177.500	0.346

BMI: Body-mass index, HDL: High-density lipoprotein cholesterol, LDL: Low-density lipoprotein cholesterol, TG: Triglyceride, TCHOL: Total cholesterol, HbA1c: Hemoglobin A1c, IQR: Interquartile range, U: Mann–Whitney U test statistics

Table 4. Comparison of gender, smoking status, and comorbidities between control and coronary ectasia groups

Variable		Control group (%)	Coronary ectasia group (%)	χ^2	p
Gender	Male	67 (46.207)	78 (53.793)	2.406	0.121
	Female	44 (57.143)	33 (42.857)		
Smoking	Yes	44 (48.889)	46 (51.111)	0.075	0.785
	No	67 (50.758)	65 (49.242)		
DM	Yes	21 (38.889)	33 (61.111)	3.524	0.060
	No	90 (53.571)	78 (46.429)		
HT	Yes	44 (43.564)	57 (56.436)	3.070	0.080
	No	67 (55.372)	54 (44.628)		

DM: Diabetes mellitus, HT: Hypertension, χ^2: Chi-square test statistics

The bar plots indicate that patients with CAE tend to have higher weight and BMI, while height remains similar between the groups (**Figure 1**).

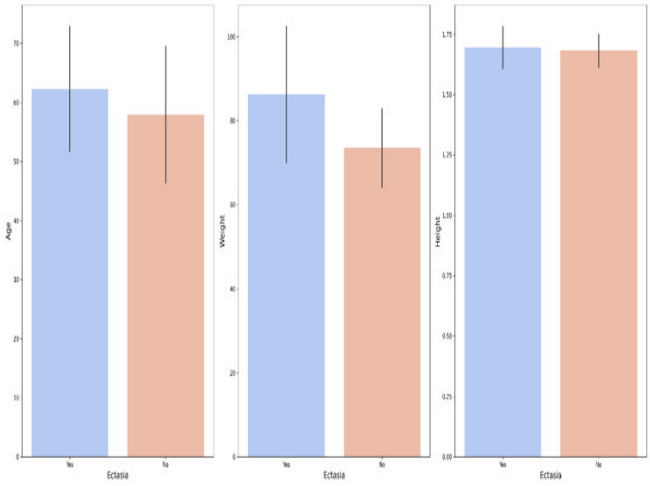

Figure 1. The bar plots coronary artery ectasia between control group

The violin plots show the distributions of BMI, HDL, LDL, TG, TCHOL, glucose, and HbA1c, indicating increased variability and elevation in BMI within the CAE group (**Figure 2**).

Overall, the results suggest that higher BMI and weight may be associated with CAE, while other metabolic parameters

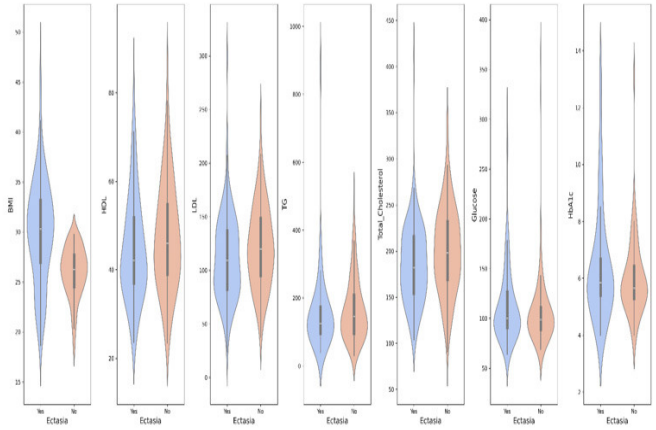

Figure 2. Violin graph showing the distribution of BMI and some blood parameters in the patient and control groups
BMI: Body-mass index, HDL: High-density lipoprotein cholesterol, LDL: Low-density lipoprotein cholesterol, TG: Triglyceride, HbA1c: Hemoglobin A1c

such as glucose and lipid profiles do not differ significantly between the groups.

The results of the Spearman correlation analysis (**Figure 3**) revealed a moderate positive correlation between CAE and BMI (ρ=0.455; p<0.001). Additionally, age (ρ=0.192; p=0.004) and TCHOL (ρ=−0.176; p=0.012) exhibited a low degree of influence. The heatmap is presented in **Figure 4**.

Spearman's Correlations

Variable		Ectasia	Gender	Smoking	DM	HT	Age	BMI	HDL	LDL	TG	TCHOL	Glucose	HbA1c
1. Ectasia	Spearman's rho	—												
	p-value	—												
2. Gender	Spearman's rho	0.104	—											
	p-value	0.122	—											
3. Smoking	Spearman's rho	0.018	0.332***	—										
	p-value	0.786	< .001	—										
4. DM	Spearman's rho	0.126	-0.116	-0.147*	—									
	p-value	0.061	0.084	0.028	—									
5. HT	Spearman's rho	0.118	-0.189**	-0.183**	0.304***	—								
	p-value	0.080	0.005	0.006	< .001	—								
6. Age	Spearman's rho	0.192**	-0.114	-0.220***	0.228***	0.232***	—							
	p-value	0.004	0.091	< .001	< .001	< .001	—							
7. BMI	Spearman's rho	0.455***	-0.002	0.039	0.213**	0.215**	0.052	—						
	p-value	< .001	0.971	0.562	0.001	0.001	0.440	—						
8. HDL	Spearman's rho	-0.118	-0.239***	-0.080	-0.059	-0.161*	0.151*	-0.150*	—					
	p-value	0.094	< .001	0.259	0.401	0.022	0.032	0.032	—					
9. LDL	Spearman's rho	-0.137	-0.195**	-0.023	-0.080	-0.095	-0.115	-0.027	0.167*	—				
	p-value	0.051	0.005	0.742	0.256	0.177	0.100	0.698	0.017	—				
10. TG	Spearman's rho	-0.087	0.002	5.115×10^{-4}	0.097	0.179*	-0.279***	0.138*	-0.365***	0.193**	—			
	p-value	0.214	0.974	0.994	0.168	0.010	< .001	0.049	< .001	0.006	—			
11. TCHOL	Spearman's rho	-0.176*	-0.214**	-0.075	-0.035	-0.054	-0.144*	-0.003	0.259***	0.860***	0.426***	—		
	p-value	0.012	0.002	0.288	0.624	0.445	0.040	0.963	< .001	< .001	< .001	—		
12. Glucose	Spearman's rho	0.078	-0.039	-0.125	0.412***	0.067	0.148*	0.191**	-0.041	-0.071	0.138	0.052	—	
	p-value	0.265	0.577	0.071	< .001	0.340	0.033	0.006	0.566	0.317	0.054	0.470	—	
13. HbA1c	Spearman's rho	0.085	-0.070	-0.117	0.630***	0.305***	0.305***	0.259**	-0.111	0.060	0.165	0.072	0.584***	—
	p-value	0.347	0.435	0.190	< .001	< .001	< .001	0.003	0.220	0.507	0.068	0.425	< .001	—

* p < .05, ** p < .01, *** p < .001

Figure 3. Spearman correlation analysis
BMI: Body-mass index, HDL: High-density lipoprotein cholesterol, LDL: Low-density lipoprotein cholesterol, TG: Triglyceride, TCHOL: Total cholesterol, HbA1c: Hemoglobin A1c, DM: Diabetes mellitus, HT: Hypertension

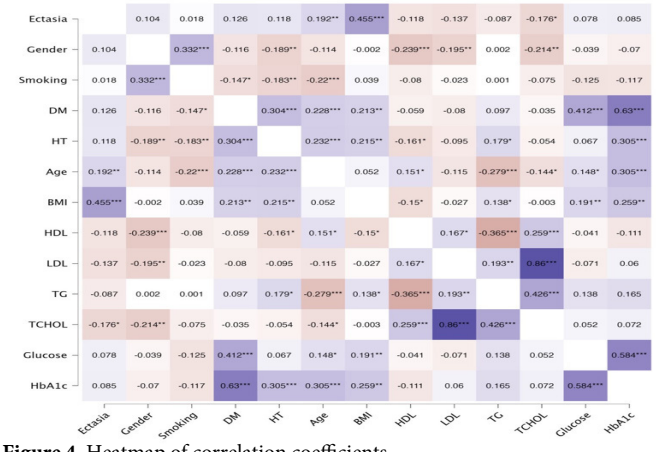

Figure 4. Heatmap of correlation coefficients
BMI: Body-mass index, HDL: High-density lipoprotein cholesterol, LDL: Low-density lipoprotein cholesterol, TG: Triglyceride, TCHOL: Total cholesterol, HbA1c: Hemoglobin A1c, DM: Diabetes mellitus, HT: Hypertension

DISCUSSION

Since BMI was much greater in CAE patients than in the control group, this study's findings point to a substantial correlation between higher BMI and CAE. The significance of BMI in the development of CAE merits more research, because obesity is a known risk factor for cardiovascular illnesses.

Obesity contributes to cardiovascular pathologies through multiple mechanisms, including endothelial dysfunction, systemic inflammation, and atherosclerotic progression. High BMI is associated with increased pro-inflammatory cytokines, oxidative stress, and impaired nitric oxide bioavailability, all of which can contribute to vascular remodeling and arterial dilation.[17,18] The underlying mechanisms of CAE remain incompletely understood; while some findings point to an atherosclerotic origin, other studies indicate a separate pathological process.[19] The moderate positive correlation found between BMI and CAE in our Spearman analysis (ρ=0.455; p<0.001) further strengthens the suggestion that obesity-related factors play a role in vascular wall changes specific to ectasia.

Obesity is considered a significant contributing factor in the development of CAE through multiple interrelated mechanisms. Increased visceral and epicardial adipose tissue (EAT) in obese individuals promotes chronic low-grade inflammation, with elevated levels of cytokines such as IL-6 and TNF-α. These proinflammatory mediators stimulate the production of matrix metalloproteinases (MMPs), which degrade the structural components of the vascular wall, including collagen and elastin, leading to arterial dilatation.[20] Additionally, obesity is associated with oxidative stress due to an excess of reactive oxygen species (ROS), which reduces nitric oxide (NO) availability and impairs endothelial function. This process further weakens vascular integrity and facilitates abnormal vascular remodeling.[21] Furthermore, EAT, which is increased in obesity, acts as a local source of inflammatory cytokines and oxidative mediators, exerting paracrine effects on adjacent coronary arteries and contributing to wall degeneration and dilation.[22] In summary, obesity contributes to the pathogenesis of CAE through inflammation, oxidative stress, endothelial dysfunction, and the proinflammatory effects of epicardial fat, ultimately leading to weakening and structural alteration of the coronary vessel wall. Although epicardial fat was not directly measured in this study, the elevated BMI and weight in CAE patients may reflect increased visceral and epicardial fat burden, suggesting a potential mechanism behind the vascular changes observed. Thus, the observed moderate association between BMI and CAE in our study reinforces the multifactorial role of obesity in coronary vessel remodeling and ectasia formation.

A study conducted in 2022 demonstrated that type 2 DM reduced the diameter and length of dilated vessels in patients with CAE. This finding suggests that diabetes has a significant impact on the pathophysiology of CAE.[23] While no statistically significant differences were observed in lipid parameters except total cholesterol in our study prior studies provide evidence suggesting otherwise. For instance, Mu et al.[24] found a significant association between remnant cholesterol and CAE (adjusted OR≈1.95, p=0.001). Qin et al.[25] identified

triglycerides and LDL/HDL ratio as independent predictors of CAE. The absence of significant findings in our study may be due to limited sample size, population-specific variations, limitations of a cross-sectional approach, and retrospective design.

Additionally, the assertion that systemic hypertension imposes chronic mechanical stress on arterial walls, facilitating vessel dilation and remodeling, is well-supported by scientific evidence.[26] While hypertension prevalence was higher in the CAE group, this difference did not reach statistical significance in our cohort. Nonetheless, its potential contribution cannot be excluded. In a study conducted by Xi et al.,[27] 36.790 patients who underwent percutaneous coronary intervention (PCI) between 2016 and 2018 were evaluated, and the prevalence of CAE was found to be 1.6%. However, the specific role of BMI as an independent risk factor for CAE remains a topic of debate. A study conducted on patients with acute myocardial infarction found that the prevalence of coronary ectasia was 8.5%, and that obesity and diabetes were associated with this condition. These findings suggest that obesity may play a role in the development of coronary ectasia.[28]

Obesity is associated with chronic systemic inflammation, endothelial dysfunction, and increased coagulation activity, all of which contribute to coronary microvascular impairment.[29] Given that obesity is a modifiable risk factor, its potential contribution to CAE highlights the importance of weight management and metabolic control in reducing cardiovascular risks. Future research should explore whether BMI reduction through structured interventions could influence CAE prevalence and outcomes.

Limitations

This study has some limitations. First, its retrospective design may introduce selection bias, and a prospective cohort study would provide more robust causal inferences. Second, while BMI is widely used as an obesity indicator, it does not differentiate between adipose tissue distribution and lean body mass, which might affect cardiovascular risk stratification. Further studies incorporating waist-to-hip ratio, visceral adiposity measures, and inflammatory biomarkers could offer deeper insights into the mechanistic relationship between obesity and CAE.

As inflammatory biomarkers were not available in our dataset, they were not included in the scope of this study. Due to the retrospective design of our study, alternative anthropometric measures such as the waist-to-hip ratio could not be included. Finally, genetic predisposition and inflammatory markers should be evaluated in future studies to better understand the molecular underpinnings of CAE development.

CONCLUSION

In this study, we evaluated the relationship between BMI and CAE by comparing the demographic, metabolic, and lipid parameters of patients with CAE and a control group. The findings indicate that patients with CAE have significantly higher BMI and weight compared to the control group, suggesting a potential association between increased body mass and the presence of CAE.

Although there were no significant differences in lipid profiles, glucose levels, or HbA1c between the two groups, the higher prevalence of DM and HT in the CAE group may indicate a possible link between metabolic disorders and the development of CAE. However, these associations did not reach statistical significance.

These results suggest that higher BMI may be a contributing factor to the development of CAE, potentially due to its role in systemic inflammation, endothelial dysfunction, and vascular remodeling. Further prospective studies with larger sample sizes are needed to confirm this relationship and to explore the underlying mechanisms linking BMI and CAE.

ETHICAL DECLARATIONS

Ethics Committee Approval

The study was conducted with the permission of Bolu Abant İzzet Baysal University Non-interventional Clinical Researches Ethics Committee (Date: 21.01.2025, Decision No: 2025/23).

Informed Consent

Because the study was designed retrospectively, no written informed consent form was obtained from patients.

Referee Evaluation Process

Externally peer-reviewed.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

Financial Disclosure

The authors declared that this study has received no financial support.

Author Contributions

All of the authors declare that they have all participated in the design, execution, and analysis of the paper, and that they have approved the final version.

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